Craniocerebral Injuries

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THE PURPOSE of this presentation is to review again the major types of injuries to the brain, the important symptoms and the danger signals, and the recent developments in management.

Cerebral concussion is the least serious of brain injuries. There is relatively little injury to the brain, and often the patient does not lose consciousness. When examined shortly after the concussion takes place the patient will be oriented and no pathologic-neurologic signs will be present. There may be innumerable subjective complaints, but the majority of patients make a complete and rapid recovery.^{1, 13}

The postconcussion syndrome is a definite entity, and the complaints are due to injury. Three definite factors contribute to the symptomatology referred to as the postconcussion syndrome.

- 1. Persistent headache, often beginning in the suboccipital area and radiating forward, is common in patients who have had concussion. It is associated with tenderness and a variable degree of spasm of the suboccipital muscles. In addition, pain is evoked by palpation over the greater occipital nerve, on one or both sides. These pains are due to the associated sprain of the cervical spine with resultant radiculitis of the upper cervical roots.^{4, 10} Rarely is any abnormality noted in spinal x-ray films.
- 2. Dizziness and feeling of instability is most often present in patients who struck the occiput against a solid object in a fall backward.⁶

Persons falling backward are unable to utilize the postural, labyrinthine and optical righting reflexes, and the result is more severe trauma to the vestibular and labyrinthine apparatus. After concussion of that kind there may be variable disturbance in balance and gait closely akin to that associated with labyrinthitis.⁹

3. The third factor in "postconcussion syndrome," characterized by lightheadedness, chronic fatigue, insomnia, restlessness and a feeling of inadequacy and insecurity, is usually noted in patients with lesser injuries and usually without even a period of unconsciousness. This group of symptoms is occasioned by a fright reaction, followed by the exhaustion syndrome as originally described by Seele. This is best illustrated and contrasted by citing two kinds of trauma: One kind is severe

- The postconcussion syndrome is a definite entity, and the patient's complaints are due to legitimate injury. The following three factors contribute to the symptomatology:
- Headaches, caused by the associated sprain of the cervical spine with resultant radiculitis giving rise to muscle spasm and subeccipital headaches.
- 2. Vertigo, due to trauma of the vestibular and labyrinthine apparatus.
- 3. A vasomotor imbalance and instability resulting from the reaction to "stress."

In treatment of an unconscious patient great care must be taken to prevent anoxia and to balance the fluids and electrolytes.

If coma develops following brain injury, hemorrhage into the cerebellar fossa as well as above the tentorium must be considered.

injury with prolonged unconsciousness; the other is the trauma of narrow escape. The patient with the severe injury and prolonged unconsciousness does not even remember the accident and makes prompt and complete recovery, while the man who has had a narrow escape may dwell on the details of the near-calamity until he becomes invalided.

These factors in the production of the postconcussion state are of great importance. They are points of contention in the medico-legal aspects of the subject. Physicians often become involved when requested to express expert medical opinion as to the extent of injury in such circumstances, for the patient often is accused of "litigation neurosis."

Newspapers carry many accounts of death caused by a fractured skull. Yet no one has ever died of a fracture of the skull. Indeed, "skull fracture" is a term so often misinterpreted that it should be abolished, for misunderstandings of its full significance (or lack of it) leads indirectly to thousands of deaths a year in the United States. A patient with fractured skull may have only a minor injury to the brain, whereas a severe contusion, and especially injury to the brain stem, often occurs without fracture of the skull.11 The misunderstanding as to the meaning of "skull fracture" and as to the significance of the condition stems from erroneous interpretation as to the cause of death as expressed by the "autopsy surgeon." The misdiagnosis at autopsy is very serious, for it teaches the average physician to seek for x-ray evidence of fracture and, when none is found, "he, as well as the patient's

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family, is lulled into a false sense of security about the patient's condition and degree of his injuries."8

Postmortem examination of an injured brain must include sections through the brain stem, and, if necessary, histopathologic study of specimens.

Of course a linear fracture crossing the meningeal groove in the temporal area, a condition more often seen in association with less severe injury of a child, may tear the meningeal artery and bring about epidural hemorrhage within a few hours. The roent-genologic technique of the physician who first examines a patient may be inadequate to show a break of this kind, or he may not have the special experience or knowledge needed to evaluate it properly.

Occasionally, fracture of the skull involves a paranasal sinus, and the development of infection—or, less commonly, of intracranial aerocele—then is a possibility.

A far more dangerous injury than fracture of the calvarium is the absence of fracture in an injury that causes violent movement of the entire cranial contents, either by the mechanism of "skull deformation, brain displacement or transmitted waves of force through the brain." When the skull is fractured by an object striking it, the force of the blow is to a degree spent in the fracturing, and thereby lessens the force of movement transmitted to the entire calvarium.

Cerebral contusion presents a problem in careful management of an unconscious patient—once the diagnosis is established. Hemorrhage requires exact diagnosis and prompt surgical intervention.

Following is a case report which will emphasize the ever-changing development and capriciousness in the progress of a severe head injury and indicate the steps to be taken for survival of the patient.

A man 62 years of age was thrown from a moving vehicle during a collision. He landed some 30 feet away, his head striking a curbstone. When examined, two and a half hours after injury, he was in deep coma; scalp lacerations were present over the right parietal area; blood and cerebrospinal fluid were leaking from the right ear; a local contusion was present over the anterior chest wall and definite crepitus was palpable over that area. The patient was lying on his back and the tongue partially obstructed breathing and sometimes stopped it for a moment. Breathing would then resume with a forceful expulsion resembling (to the uninitiated) Cheyne-Stokes respiration.

The temperature was 96° F rectally. The skin was cold; pulse rate 96 and blood pressure 98/70 mm. of mercury. Upon neurological examination, peripheral facial paralysis on the right was noted. There was motor weakness of the left arm and hyperreflexia in both the left leg and left arm. Babinski's

sign was present on the left. The pupils were small and equal.

The diagnosis was cerebral contusion, compound skull break with spinal fluid leak, crush injury of the chest and shock. Treatment consisted of warm blankets, infusion of 5 per cent glucose solution, the placement of an adequate airway and occasional nasopharyngeal suction. The patient was placed in an oxygen tent. The abdomen was soft. Catheterization yielded 250 cc. of clear amber urine.

The patient was not disturbed for spinal punctures or x-ray studies. The scalp was repaired the following morning when shock had abated. A plain film of the chest was taken and several fractures of ribs on the right side were observed. The patient remained unconscious for eight days.

Feeding was carried out through a nasal tube. There was gradual improvement and the strength returned in the upper left extremity. Roentgenographic studies of the skull then revealed an extensive linear fracture extending from the right parietal area downward into the base and passing through the internal acoustic meatus. The pineal shadow was not visible. Fracture of the right acromioclavicular joint, fracture of the carpal bones in the right hand, and fracture of the right clavicle were also observed roentgenographically.

The patient gradually improved and became ambulatory in six weeks. Babinski's sign was no longer present. Although alert, the patient was still confused and disoriented. A spinal puncture was done. The pressure was 200 mm. of water and the fluid was faintly xanthochromic. No abnormality was noted in a fundoscopic examination. Reflexes were still hyperactive on the left. Although there remained some suspicion of subdural accumulation of blood, in view of his general improved condition the patient was permitted to go home, still under careful observation. Ten days later, he became confused and began dragging his left leg. Upon examination, definite motor weakness of the left upper and lower extremity was noted, and Babinski's sign was present. The pupils remained equal and upon fundoscopic examination no evidence was seen of increased intracranial pressure. An electroencephalogram showed amplitude asymmetry over the left hemisphere. At operation an encapsulated subdural hematoma was removed from over the left hemisphere. The patient eventually recovered.

This case illustrates the following principles, which have been stressed by other observers:

The Treatment of Shock

Rarely is shock associated with injuries of the brain alone, and careful examination should always be made for injuries of the chest and abdominal viscera.² Most frequent of these are: Rupture of the spleen in upper abdominal injuries and rupture of the urinary bladder, prevalent especially in motorcycle accidents. A simple procedure often overlooked is diagnostic catheterization.

Careful observation will often reduce the amount of handling of a critically injured patient. It is wise to refrain from disturbing the patient for roentgen studies and spinal puncture until he has rested and his condition has stabilized.

The Prevention of Anoxia

An open respiratory passage for adequate oxygenation of the brain is mandatory for a patient in coma. A deeply comatose patient, often lying on his back, may aspirate blood, mucus or vomitus and the anoxemia caused by inadequate respiratory exchange may do more harm to the brain than was done by the original injury. Echols³ expressed belief that cerebral edema in such cases is caused by anoxia rather than by the cerebral injury. One should not hesitate, therefore, to perform trache-otomy in a patient in the condition described.

Fluid and Electrolyte Balance

Fluid balance must, of course, be maintained by administration of about 2,500 cc. of fluid in a 24-hour period. Better yet, a nasal stomach tube is an excellent way of providing the patient with frequent small feedings; in this way, the protein, fluid and electrolyte needs, as well as vitamin therapy, may easily be supplied.

Hematomas

The various types of hemorrhage must be kept in mind. An epidural (meningeal) hematoma or a subdural hemorrhage usually develops within several hours after the injury, while a chronic subdural hematoma may take several weeks to develop.

It is well to remember that in a patient unconscious from cerebral contusion, hematoma may develop without the classical feature of a lucid interval, since the long period of coma associated with the severe injury overlaps the lucid interval period.⁵ The development of a hemorrhage requiring surgical intervention may be suspected when an unconscious patient becomes more restless, has sudden temporary slowing of the pulse, increase in pulse pressure and/or aggravation of focal or localizing motor signs.

While negative explorations are necessarily rare, bilateral trephine opening is a wise measure when in serious doubt, especially since subdural hematoma is often bilateral.

Valuable aids in diagnosing the surgical hematoma are x-ray films of the skull for possible visualization of a pineal shift, and angiography and/or ventriculography.

Hematomas of the Posterior Fossa

The same factors that give rise to bleeding in the anterior fossa may also prevail in the posterior fossa. Hence hemorrhage may be epidural, subdural or intracerebellar.

There is still another lesion in the posterior fossa that follows trauma — adhesive arachnoiditis over the cerebellum and brain stem, sealing over the cisterna magna and over the basal cisterns, thus preventing escape of the ventricular fluid.¹⁴ The cisterna magna then dilates, forming an arachnoidal cyst. The cerebellar lobes become separated and the tonsils are forced downward into the spinal canal. The resultant blocking of the ventricular system causes acute intracranial pressure, and pain may be distributed over the roots of the upper cervical spinal cord.

The author has operated on three patients with a lesion of this type. Two were adults, aged 33 and 46, and one an infant three and a half months of age with developing hydrocephalus. All had definite history of injury.

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